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Diet and Nutritional Factors in the Aetiology of Colon Cancer *(Review)*

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Abstract. *Epidemiological studies suggest that diet may be a major risk factor in the aetiology of colon cancer. Total fat, meat, animal protein and dietary fibre have received most attention. A high-fat – high-protein diet leading to increased faecal steroids has been implicated, but no active carcinogen derived from these has been isolated from human faeces. Alternatively, a refined, low fibre diet with decreased faecal bulk leads to increased large bowel concentration of carcinogens of co-carcinogens which contribute to the development of a malignant tumour. The aetiology of colonic cancer is probably multifactorial but a diet low in animal fat and protein and abundant in vegetables containing vitamin A and lignans may protect against colon cancer.*

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Introduction

Cancer of the colon and rectum may be the most important malignant disease in the western world. It is a major cause of cancer mortality, being second only to lung cancer in men and the third leading malignancy in women (1, 2). The American Cancer Society estimated that about half a million of the current population of the United States have been diagnosed as having colorectal cancer, accounting for more than 130,000 new cases each year with almost 60,000 deaths being attributed to this type of cancer (1-5).

Epidemiological studies have clearly demonstrated that several types of human cancers relate to identifiable causes (4) and that as much as between 70% and 90% of all cancers are related to our environment and lifestyles; more specifically, about 40% to 60% of environmental cancers are linked in some way to diet and nutrition (3). Diet is considered to be the most important risk factor for cancer, ranking second after tobacco (2), although it is not clear which specific dietary component or components cause cancer.

The worldwide incidence of colon cancer is highest in the United States, West Germany, Australia and New Zealand; and colon cancer is the second most common neoplasm in England, Wales, Sweden, Denmark and many other Western European countries. It is however rare in Africa, Asia and South America (2, 5, 6). Studies of the aetiology of colon cancer indicate that diets particularly high in total fat and meat, and low in dietary fibre (vegetables and micronutrients), are generally associated with an increased incidence of colon cancer in man (2, 3, 5-8).

Studies of migrants with respect to colon cancer have demonstrated that migrants from low incidence to high incidence areas acquired a higher risk for colon cancer, suggesting that the risk of developing colon cancer is affected by the place to which the individual has moved rather than the place from which he came (9, 10). The last decade has produced evidence that certain population groups, whose lifestyle habits differ considerably from those of the general populations, have lower than expected incidence.

Suggested hypotheses

Several hypotheses have been put forward concerning diet and the aetiology of colon cancer. One of these, sometimes

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referred to as fibre theory, is due to Burkitt (5) who suggested that populations consuming a diet rich in fibre have a low incidence of large bowel cancer, while those eating a refined diet with little fibre have a higher incidence. He argued that large bowel tumours are related to factors characteristic of modern Western society, and that a refined diet low in fibre, through a decrease in faecal bulk and transit time, leads to an increased concentration of carcinogens or other substances contributing to the development of a malignant tumour.

Wynder *et al* (11) proposed that colon cancer incidence is mainly associated with dietary fat and meat, and suggested that fat influences the composition of the faecal flora and in this way may be involved in the pathogenesis of colon cancer. Hill and his group (6) have postulated a role for gut bacteria, in particular certain clostridia, and suggested that colonic cancer is caused by metabolites produced in the colon from some benign substrate by the bacterial flora that act as carcinogens or cocarcinogens in colonic carcinogenesis. Since diet controls the concentration of the substrate, *eg* faecal steroids, and the composition and metabolic activity of the flora, it would consequently determine the concentration of the carcinogen or cocarcinogen produced (18).

Epidemiology of colon cancer

During the past decade, cancer of the colon has been the subject of several different types of epidemiological reviews (5, 9, 10, 13, 15) which have investigated the influence of environmental and nutritional factors on the occurrence of cancer of the large bowel.

Variation of the incidence of colon cancer between countries. The incidence of colon cancer varies approximately twenty-fold or more from one part of the world to another. The frequency is greater in the developed countries, with the exception of Japan. The highest incidence rates are reported in North America, Australia, New Zealand and Western Europe, with the exception of Finland where the incidence is one of the lowest in the developed countries. Intermediate rates are found in Eastern Europe, while the lowest rates are observed in Asia, Africa and South America (2, 3, 5 11).

There are substantial regional variations: for example, the incidence is three times higher in Denmark than in Finland, and in Sweden there is a graduated increase in the incidence from north to south. A similar occurrence is seen in India, where there is a low incidence amongst the Punjabis in the north compared to south Indians (12).

Distinction between cancers of the colon and rectum. The International Classification of Diseases separates cancer of the colon and rectum (13). The highest incidence of colon cancer is found in the United States, whereas the regions at highest risk for rectal cancer are situated in Western Europe (13, 14).

Sex and age incidence. Data in Table I (13) show there is a difference in the sex ratios for cancer of the colon and rectum; rectum cancer is more common among males, whereas colon cancer is seen in both sexes at similar rates.

Demographic characteristics

Urban and rural differences / time trends. Cancer of the colon occurs in general more frequently in urban than in rural areas. Studies of time trends show that for both sexes colon cancer incidence increases gradually in almost all countries. The increase in incidence is more marked in low risk areas but the pattern observed for rectum cancer is less uniform (15).

Socioeconomic status. In general, cancer of the colon and rectum is a disease of economically developed countries, with the exception of Japan and Finland. Recent studies in Cali, Columbia indicate that the risk of this cancer is four times greater in the upper than in the lower socioeconomic classes for both sexes and in all age groups (14). The data from England and Wales suggest a tendency to higher risk for males in the upper social classes. The higher prevalence amongst the upper social class is also noted in Hong Kong (4) and in Japan (3).

Religion and lifestyle. In the United States, mortality for large bowel cancer in the Seventh Day Adventists (SDA) living in California is substantially lower (60%) than among the general population and a sample of non-SDA who were demographically similar to SDA (10). It is claimed that the SDA traditionally consume virtually no meat, they follow a lacto-ovo-vegetarian diet, containing no poultry or fish but including milk products and eggs.

Similarly, the incidence of large bowel cancer is lower in Mormons than in other white population groups in the United States (10). Although the Mormons consume meat, they eat more whole grain breads and cereals.

In Bombay, the various religious groups have contrasting incidence rates of cancer of colon and rectum (15). The Parsees who are a meat consuming religious group exhibit higher incidence than the Hindus who are generally lacto-vegetarian.

Racial differences. The data from cancer registers (16) show that in Hawaii colon cancer rates among Hawaiians and Filipinos are low compared to those for Caucasians, Japanese or Chinese. In the San Francisco Bay area, however, rates show little variations between whites, blacks, Chinese and Japanese. Further evidence of racial differences in the incidence of large bowel cancer comes from New Zealand with a two- to threefold difference between non-Maoris and Maoris, or from Singapore where the incidence is three times higher in Chinese than in Malayans or Indians. These observations once more suggest the important role of different lifestyles (13).

Dietary factors associated with colon cancer

There are several dietary and nutritional factors that are postulated to play predominant roles in colon carcinogenesis

Table I. *Cumulative incidence rates, 0-74 years (per cent) for colorectal cancers by sex for selected cancer registers.*

	Males	Females
Canada, Quebec	3.9	3.3
United States, Connecticut	5.9	4.3
United States, Seattle	4.8	3.9
Columbia, Cali	1.0	0.9
India, Bombay	0.8	0.6
Japan, Osaka	1.8	1.2
Senegal, Dakar	0.3	0.2
Denmark	4.1	3.4
Germany (FRG), Sarland	4.5	3.4
Finland	1.9	1.7
France, Cote-d'Or	4.6	2.8
Italy, Varese	4.3	2.9
Norway	3.0	2.5
Poland, Cracovia	2.7	1.6
Switzerland, Geneva	4.7	2.8
United Kingdom, Birmingham	3.8	2.8
Yugoslavia, Slovenia	2.2	1.7
Australia, New South West	4.0	3.0

From Faivre *et al* (13)

Table II. *Dietary factors associated with large bowel cancer.*

Dietary factor	Risk of cancer	Reference
Meat	Increased risk	Armstrong, Doll (14) Haenszel *et al* (30) Howell (32) Bjelke (40)
(Beef)		Haenszel *et al* (30)
	No association	Breslow, Enstrom (20) Haenszel *et al* (33) Higginson (58) Modan *et al* (59)
Animal protein	Increased risk	Armstrong, Doll (14) Jain (21) Haenszel *et al* (30)
Fat	Increased risk	Gregor *et al* (31) Wynder *et al* (3) Wynder *et al* (11) Drasar, Irving (26) Hanszel *et al* (30) Howell (32)
	No association	Graham *et al* (22) Higginson (58)
Cholestrol	Increased risk	Haenszel *et al* (30) Jain *et al* (21) Broitman (35) Liu *et al* (36)
Eggs	Increased risk	Howell (32)
Milk	Increased risk	Howell (32)
Sugar	Increased risk	Howell (32) Bristol, Heaton (57)
Fibre	Decreased risk (protective)	Burkitt (5) Reddy (10) Armstrong, Doll (14) Burkitt *et al* (54) Modan *et al* (59)
Fibre	No association	Jain *et al* (21) Miller *et al* (39) Higginson (58)
Cereals	Decreased risk (protective)	Armstrong, Doll (14) Howell (32)
Legumes	Decreased risk (protective)	Howell (32)
Vegetables	Decreased risk (protective)	Graham *et al* (22) Haenszel *et al* (30) Modan *et al* (59)
	No association	Wynder *et al* (11) Haenszel *et al* (33)
Alcohol (Beer)	Increased risk	Wynder *et al* (11) Breslow, Enstrom (20) Enstrom (34)
	No association	Graham *et al* (22) Miller *et al* (39) Higginson (58)
Vitamin A	Decreased risk (protective)	Gregor *et al* (31) Bjelke (40)
Selenium	Decreased risk (protective)	Jensen (8) Schrauzer (61)

or modifiers of cancer risk; these are outlined in Table II.

The refined calori-dense, animal protein-rich, high-fat diet of the western cultures has attracted particular attention as a possible cause. Internationally, colon cancer is strongly positively correlated with the intake of fat and animal protein, meat particularly beef, but negatively correlated with the consumption of fibre (11). Several studies show that fibre in the diet has a protective role against colon cancer. However, in case control studies the role of diet was very much less clear cut for these types of cancer with these cancer sites (18).

Several other factors have been suspected to contribute to large bowel carcinogenesis or to modify this process. Protein metabolites, such as ammonia and tryptophan, N-nitroso compounds, nitrosamines, and products derived from the surfaces of grilled meat and fish have been thought to have carcinogenic properties for the human large bowel (18, 19). Beer has been suggested as a high risk factor for rectal cancer (20). In other studies low levels of serum cholesterol have been correlated with colonic cancer and the protective role of certain vitamins such as Vitamin A has been suggested to have an inverse relationship to colon cancer (8). It has been claimed that parity could influence the occurrence of colon cancer (9). Cigarette smoking has also been suggested as a high risk factor for large bowel cancer. The results of some studies have been interpreted to suggest the protective role of substances like indoles, selenium, ascorbic acid, retinoids and some others on colon carcinogenesis in humans (9, 22).

Lipids, fats and cholesterol

Of all the dietary factors that have been associated with colon carcinogenesis, fat has probably been studied most thoroughly and produced the strongest associations. A worldwide correlation between the incidence of colon cancer and total fat consumption has been established (23). A major portion of the fat in the western diet comes from meat, and beef is believed to contribute significantly to the overall fat intake; this makes it more difficult to attribute these associations to fat intake *per se* with absolute certainty (24).

Most epidemiological, correlational and case control studies show an association between increased risk of large bowel cancer and higher fat intake; however, a few case control studies on large bowel cancer fail to show an association. The possible reasons for these discrepancies have been discussed (3).

Animal studies have shown that dietary lipids influence tumorigenesis, especially of the colon. An increase in fat intake increases tumour incidence in various tissues; conversely, animals consuming low fat diets have a lower tumour incidence. When the intake of total fat is low, polyunsaturated fats appear to be more effective than saturated fats in enhancing tumorigenesis (25).

Wynder and Shigematsu proposed that colon cancer is associated with dietary fat and suggested that fat influences the composition of faecal flora and this is involved in the pathogenesis of cancer of the colon (11).

Drasar and Irving (26) compared dietary data and the incidence of colon cancer from 37 countries, and showed that the incidence of colon cancer was highly correlated with intake of combined fat and animal protein. Armstrong and Doll (14) reported direct correlations between colon and rectal cancer incidence and mortality and *per capita* intake of total fat, based on international data. Wynder *et al* (27) suggested a correlation between the westernisation of the Japanese diet and colon cancer, significant changes being in the increase in the consumption of fat and meat. Another study on the Jewish population of New York, who eat more animal fat, showed them to have a higher rate of colon cancer than other New York residents; while the seventh Day Adventists, who eat very little or no meat *(ie* ovo-lacto-vegetarians) and who have a lower intake of saturated fats than most Americans, had a somewhat lower mortality rate for colon cancer.

A number of investigators have come up with contrasting results. For example, in spite of a high dietary fat intake and a parallel high incidence of coronary heart disease, the incidence of colon cancer is relatively low in Finland. In a recent International Agency for Research on Cancer (IARC) coordinated study (28), which included population samples from Helsinki and rural Finland, and from Copenhagen and rural Denmark, no trends paralleling the colon and rectal cancer incidences emerged with regard to average daily fat and meat intake. Bingham *et al* (29) found no significant association of fat intake with mortality from colon and rectal cancers in Britain.

Meat - animal protein. The consumption of meat, which forms an important source of dietary fat, has been related to colon cancer in a number of large scale studies (Table II).

In their case control studies on large bowel cancer among Hawaiian Japanese, Haenszel *et al* (30) found that of all the different meats investigated, beef consumption produced the most pronounced case control differences. However, another case control study in Japan and a cohort study revealed a negative association of meat intake with the risk of colon cancer (9).

Gregor (31) correlated data from 28 countries and found an association between the rate of increase in the incidence of colon cancer and an increase in the consumption of animal protein. Their data supported a promoter role for diet during the development of the disease rather than a role in its initiation. In a more extensive study, Drasar and Irving (26) confirmed the close association of colon cancer with the intake of meat and also with that of combined fat, consisting mainly of fat derived from meat. This association has later been demonstrated in similar studies of internationally available data (14)

Howell (32) reported a high correlation between colon cancer mortality and meat intake, particularly beef consumption based on international *per capita* intake data.

The association between consumption of meat, especially beef, and large bowel cancer (30) was not reproduced in studies conducted in Buffalo, New york (22) and in Japan (33), nor in parallel cohorts followed prospectively in Minnesota and Norway (4). Furthermore, Enstrom (34) also noted that trends in beef intake in the United States do not correlate with trends in the incidence of and mortality from colorectal cancer.

Cholesterol and colon carcinogenesis

Cholesterol has been implicated as an aetiological factor in both arterial and neoplastic disease (35). In general, epidemiological evidence suggests that populations consuming a high cholesterol high fat diet tend to be more prone to certain types of malignancies, such as colorectal and breast, than those who consume low to moderate amounts of these two constituents.

Dietary cholesterol and faecal cholesterol. It has been proposed recently that faecal cholesterol increases the risk of large bowel cancer (8) and that there is a strong international correlation between colon cancer mortality and dietary cholesterol (36). The risk of colon cancer is increased in the Japanese migrants to the United States who also showed an increase in the dietary fat and cholesterol intake (37, 38). Miller *et al* (39) showed that a stepwise increase in the risk of colorectal cancer was associated with an increase in cholesterol intake.

Serum cholesterol. Several studies of serum cholesterol levels have shown an inverse relationship with colon cancer (4, 40, 41). The Framingham Heart Study (42) showed a similar relationship in men but not in women. A large study in Georgia of 3,000 cases (43) and the Honolulu Heart Study of 7,961 cases (44) showed that serum levels of cholesterol were directly related to mortality from coronary heart disease but inversely associated with colon cancer. In contrast, however, serum cholesterol was not associated with colon cancer in Chicago men and in the Yugoslavia study (45).

It appears there is conflicting evidence that increased risk of colon cancer is associated with dietary cholesterol, but also with very low serum cholesterol levels. A possible explanation might be that a high intake of cholesterol or dietary fat by persons whose metabolism maintains low serum cholesterol results in reduced biosynthesis of cholesterol and a high rate of excretion for cholesterol breakdown products in the intestines. These breakdown products could serve as substrates for the intraluminal production of carcinogens by intestinal bacteria (6, 46).

Role of dietary fibre in colon cancer

Dietary fibre has been intensively studied in relation to colon cancer. Early in 1956, Cleave (47) suggested that many of the diseases in affluent western societies were due to the depletion of fibre from the diet. Malhotra (12) noticed the differences in colon cancer incidence among northern and southern populations in India, and found vegetable fibres were abundant in the stools of Punjabis from the north, but completely absent in samples obtained from the inhabitants of Southern India.

Burkitt (5) advanced the Cleave theory and attributed the high incidence of colon cancer in certain countries to a lack of fibre in the diet, basing his hypothesis on the effects of unabsorbable fibre on the physiology of the large bowel. He argued that large bowel tumours are related to factors characteristic of modern Western society whereby intestinal transit time is slowed, small firm stools are produced and the faecal flora is altered. Slower transit would allow more time for gut bacteria to degrade intraluminal components, produce carcinogens or co-carcinogens and enable such carcinogens to act. This has been postulated to be due to the diets of western populations which differ in several respects, including large amounts of animal protein, meat, fat, sugar and highly refined cereal products but reduced amounts of fibre.

Correlation studies. International correlation studies show an inverse relation between colon cancer and dietary fibre consumption (14). The results of the IARC co-ordinated study in Copenhagen and Kuopio (28), which compared the low risk populations of Kuopio with those at high risk in Copenhagen, produced evidence for the role of dietary fibre in the prevention of large bowel cancer. These results were later supported by the findings of Bingham *et al* (29) who also showed that the intake of pentosan fraction of total dietary fibre and of vegetables other than potatoes was inversely correlated with mortality from colon cancer. In the more recent IARC co-ordinated Finnish-Danish study (48), an inverse trend with large bowel cancer was found for cereals and total dietary fibre but not for pentosans.

Some correlation studies have not supported the hypothesis that fibre intake is inversely related to the risk of colon cancer (36, 49). Lyon and Sorenson (50) found little difference in fibre intake between a low risk area and the general population of the United States.

Case control studies. A number of case control studies have shown a correlation between westernisation of the diet to include a higher content of fat and meat and a lower content of fibre, and colon cancer (11, 40, 48). Most of the studies showed that vegetables were eaten less frequently by colon cancer patients. Similarly, Graham *et al* (22) found that vegetables such as cabbage, broccoli and brussel sprouts were associated with low risk, while diets with a high intake of fat and meat, and low in fibre and vegetables, are generally associated with a high risk of large bowel cancer. The Japanese prospective study (51) also reported results comparable with the fibre hypothesis.

Protective role of dietary fibre against colon cancer

Dietary fibre is the part of ingested plant material resistant to digestive enzymes. It comprises carbohydrates, cellulose, hemicellulose, pectin and non-carbohydrate substance lignin. It is suggested that the protective effect against colon carcinogenesis may be due to adsorption, dilution or metabolism of carcinogens, co-carcinogens, and promoters of carcinogens by the components of the fibre (10).

There is evidence to suggest that certain fibres, *eg* pectin and lignin, may act as cation exchangers or may bind bile acids, and affect the metabolism of bile salts and increase the faecal excretion of these compounds. In this manner, fibre may prevent or reduce the toxic effect of certain metabolites or carcinogens (52, 53). The solvent-like effect of fibre is believed to reduce the formation of potential tumour promoters in the colon and to dilute potential carcinogens and co-carcinogens by its bulking effect (15). Slower intestinal transit time is generally associated with low fibre diets; and it is claimed that slower transit may lead to increased contact of luminal carcinogens or co-carcinogens with the internal mucosa (54).

The intestinal microflora is capable of converting both endogenous and exogenous agents into active luminal carcinogens. These microbes appear to be sensitive to various dietary substrates including fibre, some of which may be important in colon carcinogenesis (21, 55, 56)

Carbohydrates, sugar and the risk of colon cancer. In contrast to lipids and proteins which are the other two macronutrients in the diet, there is little evidence to support a role for carbohydrates *per se* in the aetiology of colon cancer (4). The main carbohydrates in the food include sugars, starches and cellulose. Cellulose has been considered under dietary fibre, while as far as starches are concerned, only limited data are available for colon cancer.

High intake of refined sugar in the diet has been associated with breast cancer (26), and recently Heaton's group (57) have suggested that a high intake of sugar depleted in fibre and a high fat diet predispose to the development of colorectal cancer. Other studies have related the intake of starchy foods, *eg* bread, potatoes and sugar, to cancer of other sites but not colorectal cancer. Thus there is little evidence to suggest a direct role for carbohydrates with the exclusion of fibre in carcinogenesis.

Alcohol and large bowel cancer. Studies have shown that alcohol taken in the form of spirits, beer or wine is an important risk factor for cancer of certain sites. Beer drinking has been associated with colorectal cancer, in particular rectal cancer, in a number of studies (11, 20, 34) but not in all studies (22, 39, 58).

The data available show conflicting results, some of which support the hypothesis that beer intake is associated with colorectal cancer and some of which do not. So far, no mechanism whereby alcohol or beer may influence colon carcinogenesis has been proposed (8).

Whether the risk is due to alcohol itself or some chemical in it is not clear; however, excessive intake of alcohol by cigarette smokers appears to act synergistically to increase the risk of cancer (4).

Other dietary factors and inhibitors of carcinogenesis

Some foods and compounds found in foods have been shown to exert a protective effect against colon cancer, but the mechanism is not clear. Ingestion of cabbage (59) and carotene-rich (*ie* dark green and deep yellow) vegetables and cruciferous vegetables *(ie* cabbage, broccoli, cauliflower and brussel sprouts) have been shown to have an inverse relationship with colorectal cancer (22). The risk of colorectal cancer was associated with an index of vegetable consumption in some counries but not in others (30). According to a hypothesis, a vegetarian diet may protect against colon cancer because it is high in fibre and lignans which may influence the mechanism that may be involved in colon carcinogenesis (60). There is evidence to suggest that vitamin A or foods containing it, *ie* retinol in whole milk and liver, and ß-carotene found in dark green and deep yellow vegetables, is associated with a reduced risk of colorectal cancer. These foods may have an anticarcinogenic activity, but it is difficult to distinguish the protective role of vitamin A, from that of its carotinoid precursors. Epidemiological data collected from over 20 countries showed an inverse relationship between selenium intake and colorectal cancer (61); a similar relationship and a protective effect of selenium was also observed by Salonen *et al* (62).

Carcinogens present in food

Some constituents present in normal food have been shown to exert carcinogenic activity. For example, compounds such as hydrazines, metabolites of mold (*eg* mycotoxins), metabolites of bacteria *(eg* carcinogenic nitrosamines may contaminate food and pose a potential risk of cancer.

Mutagens have been found in charred meat and fish, and smoking of food enhances the mutagenic and carcinogenic activity in foods (9). Of the vast numbers of food additives (nearly 3,000 in the US), only a few have been tested for carcinogenicity, and some chemicals find their way into food during packaging, for example vinyl chloride has been shown to be carcinogenic in humans and acrylonitrile produces cancer in animals (4).

Discussion

This review has examined diet and nutritional factors relevant to the aetiology of cancer of the colon and rectum. Epidemiological studies have shown a strong correlation between diets consumed by modern affluent societies and the incidence of colorectal cancer, but it has proved to be much more difficult to establish which, if any, of the dietary items or constituents of food is responsible for causing colon carcinogenesis.

International studies show that the incidence of cancer of the colon and rectum differs greatly between countries and to a limited extent within countries. In the course of these studies, it is clear that diet is a major factor which influences the development of colon cancer and that the disease has an external cause and in principle should be preventable. Mortality from colorectal cancer is directly associated with socioeconomic status and certain religious groups whose dietary habits differ from those of the general population, such as the Californian Seventh Day Adventists or the Mormons who follow a lacto-ovo-vegetarian diet or eat little meat and abstain from smoking and drinking, have a low incidence of large bowel cancer. In India, the Parsees who are a non-vegetarian group are at a higher risk of colon cancer than the Hindus who are generally vegetarians and are at low risk of cancer. Although there may be many differences other than dietary between these groups and the general population, these studies indicate which hypotheses should be evaluated further. At least some of these studies suggest that a lacto-vegetarian diet may have a protective role in colon carcinogenesis.

Of all the dietary factors investigated, total fat, dietary fibre, meat, animal protein and cholesterol have had most attention; of these, evidence is most suggestive for a causal relationship between total fat intake and the development of colorectal cancer. Although epidemiological studies suggest a strong inverse association between large bowel cancer and intake of dietary fibre, some case control studies have not shown such a relationship. Fibre, present in fruits, vegetables and cereals, has been shown to be protective against large-bowel cancer, and the consumption of certain vegetables, especially carotene-rich (*ie* dark green and deep yellow) vegetables and cruciferous vegetables (*eg* cabbage, broccoli, cauliflower and brussel sprouts), is associated with a reduction in the incidence of large bowel cancer. The responsible constituent or constituents in this food has not been identified on the basis of present data; if there is such an inhibiting effect on the development of cancer, present data suggest that specific components of fibre rather than total dietary fibre are most likely to be implicated. This is line with the recent hypothesis by which a vegetarian diet, abundant in lignans for example, may protect against colon cancer.

Certain food items have been examined for their ability to inhibit or enhance carcinogenesis. For example, certain foods containing vitamin A have been suggested to inhibit the development of colorectal cancer, while in another study the intake of selenium is inversely related to the incidence of colon cancer. Beer drinking in some countries has been associated with cancer of the rectum, but so far no mechanism has been suggested except that alcohol *per se* is not carcinogenic.

As for carbohydrates, with the exclusion of fibre in the diet, there is little evidence to suggest a direct role in colon carcinogenesis, but excessive carbohydrate consumption would lead to caloric excess which in turn may be implicated as a possible modifier of carcinogenesis.

On the basis of the data available at present, there is inconclusive evidence to reject the fat hypothesis, the fibre theory or the meat and animal proteins as the suspect foods in colon carcinogenesis. Several investigators have attempted to shed light on the mechanism by which diet may influence colon carcinogenesis (8, 9, 18, 63) and bile acids may function as carcinogens, co-carcinogens or promoters in colon carcinogenesis (3, 49, 64). However, no active carcinogen derived from bile acids has been isolated from human faeces.

The aetiology of colon carcinogenesis is certainly multifactorial, and although there have been advances, these have not always been consistent. Hill (49) and Doll (65) have stressed the need for a broadly based approach to reduce the multitude of causative factors and have pointed out that preventive medicine has a special role in this disease.

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